Lymph node histology pdf

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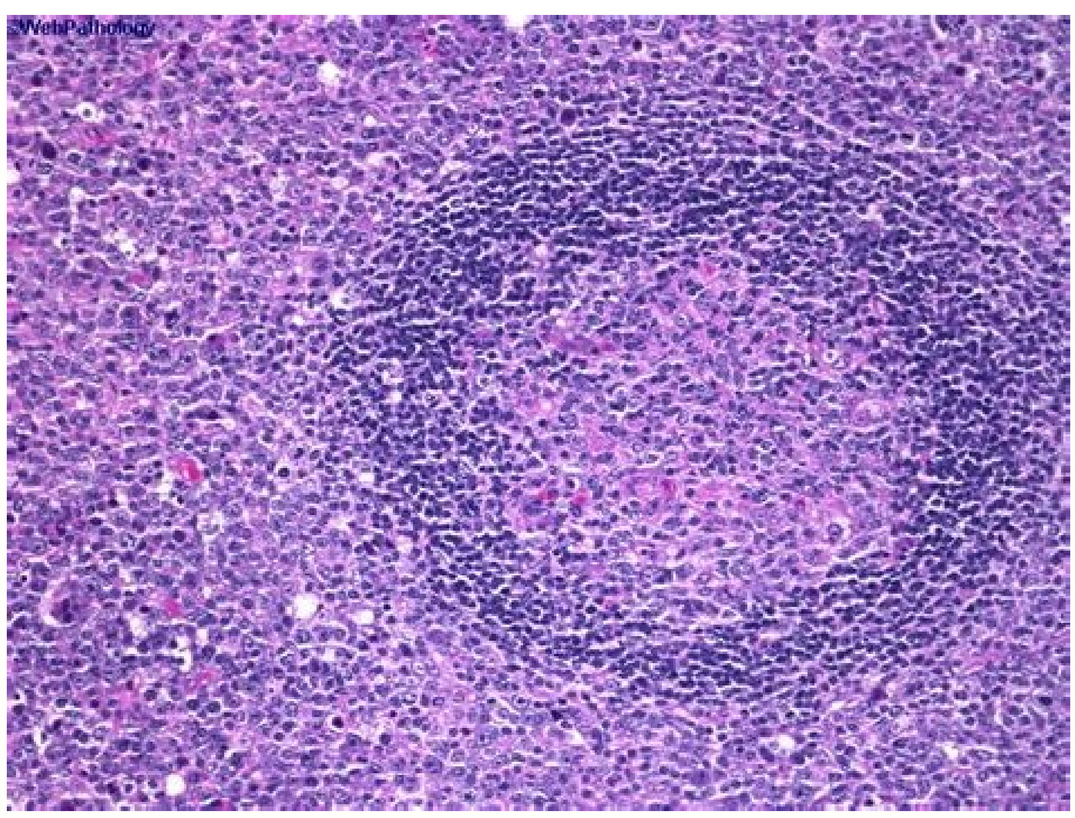

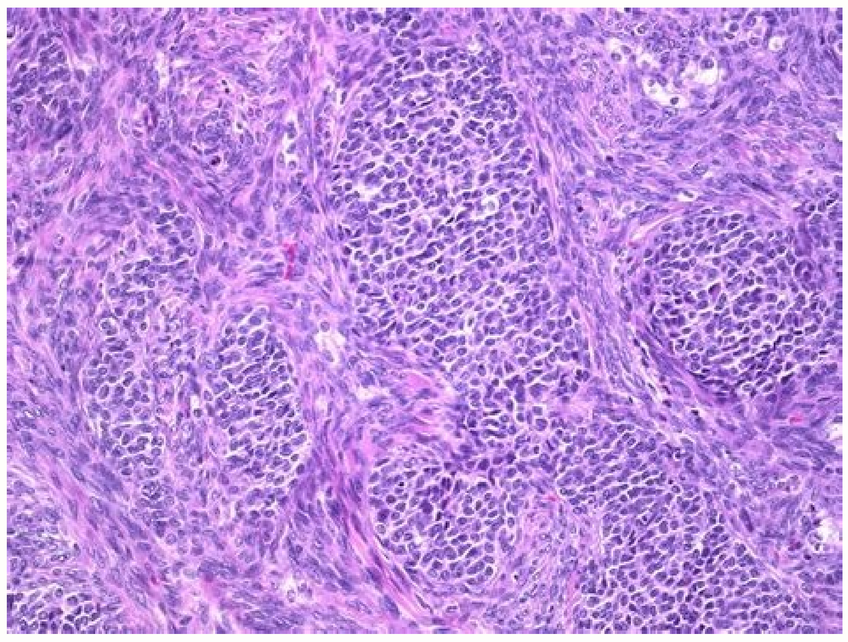

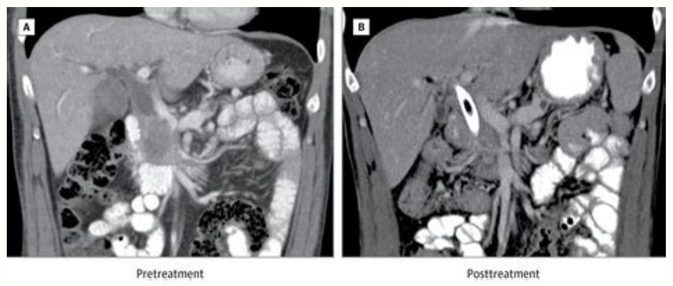

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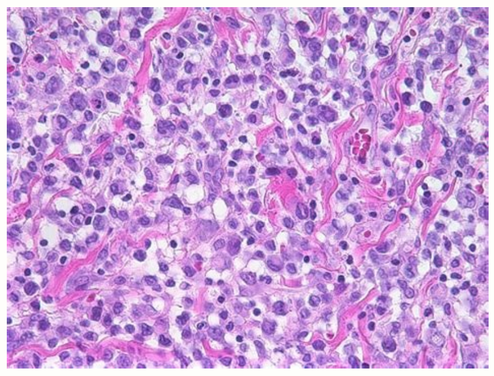

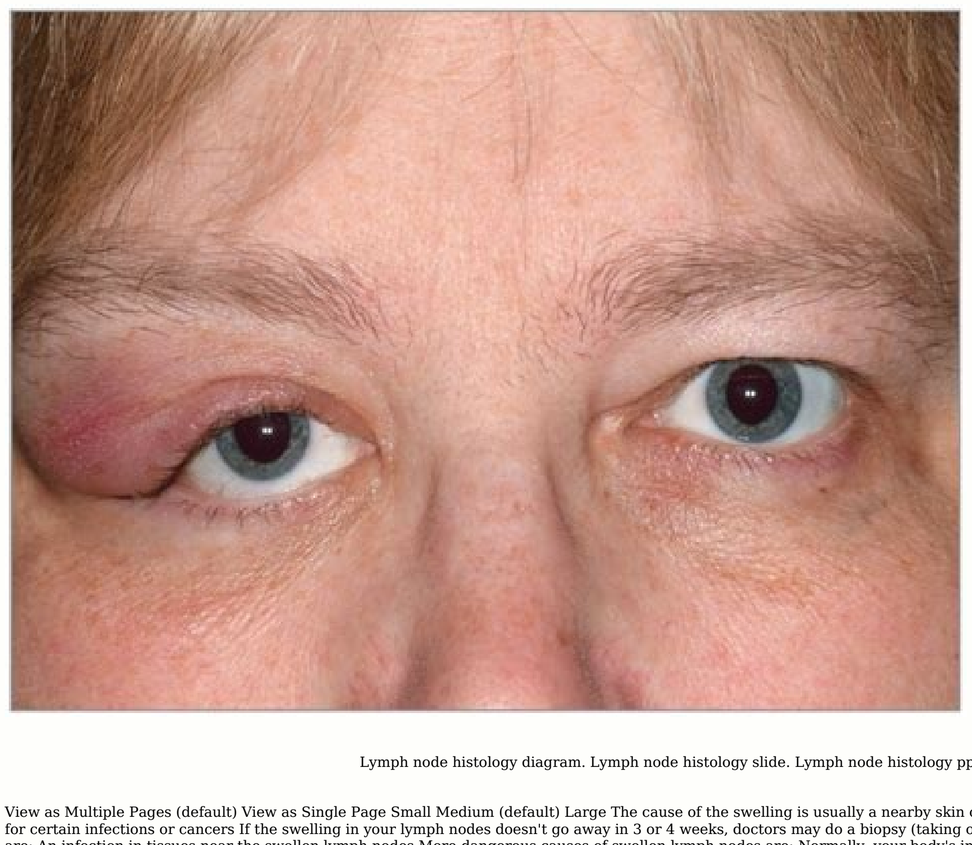

Lymph node histology diagram. Lymph node histology slide. Lymph node histology ppt. Lymph node histology labeled. Lymph node histology guide. Lymph node histology quiz. Lymph node histology pathology outlines. Lymph node histology mantle zone.

View as Multiple Pages (default) View as Single Page Small Medium (default) Large The cause of the swelling is usually a nearby skin or tissue infection or a harmless virus that goes away on its own Sometimes the cause is a more serious infection or cancer Swollen lymph nodes may hurt, or they may be painless Sometimes your doctor may do tests for certain infections or cancers If the swelling in your lymph nodes doesn't go away in 3 or 4 weeks, doctors may do a biopsy (taking out part of the tissue to look at under a microscope) People call swollen lymph nodes "swollen glands," but lymph nodes aren't really glands. There are many causes of swollen lymph nodes. The most common causes are: An infection in tissues near the swollen lymph nodes More dangerous causes of swollen lymph nodes are: Normally, your body's immune defenses kill any live germs that get into your lymph nodes. But sometimes a few germs survive and cause an infection. An infected lymph node hurts, and the skin over it turns red. Cancer cells often break off from a cancer and travel through lymph vessels to nearby lymph nodes. For example, breast cancer often spreads to the lymph nodes in the armpit that's on the same side as the cancer. Sometimes your immune defenses kill the cancer cells. But sometimes the cancer cells grow in your lymph nodes. Cancer usually makes lymph nodes very hard and stuck together. However, probably less than 1% of people with swollen lymph nodes have cancer. Not every person with swollen lymph nodes needs to go to a doctor right away. See your doctor right away if a lymph node is: Draining pus (thick, white or yellow fluid) Call your doctor if you have any of these other warning signs: The doctor will decide how quickly you need to be seen based on the warning signs and other symptoms. If you have no warning signs and you feel well, you can wait a week to see if the node returns to normal before calling your doctor. Doctors treat the cause of your swollen lymph nodes. NOTE: This is the Consumer Version. DOCTORS: CLICK HERE FOR THE PROFESSIONAL VERSION CLICK HERE FOR THE PROFESSIONAL VERSION Author: Lorenzo Crumbie MBBS, BSc • Reviewer: Jerome Goffin Last reviewed: June 29, 2022 Reading time: 22 minutes Lymph node (histological slide) The presence of foreign organisms within the blood stream can trigger a massive cascade of events that will disrupt many homeostatic microenvironments within the body. Therefore, the immune system carries out detailed surveillance of the blood in order to detect these pathogens. One method of screening takes place at the level of the lymph nodes. These are secondary lymphoid organs that are widely distributed throughout the body. Apart from its role in immune regulation, the lymphatic system is also important for immune regulation and fat absorption. Between 400 and 450 lymph nodes are scattered throughout the average human body. They are found along the lymphatic vessels, which carry fluid from the interstitial space into the main circulation. They are particularly abundant in the cervical, axillary, inguinal, perihilar, and intra-abdominal areas. These locations are vulnerable points of entry of pathogens into the host's intrinsic environment. As a result, it is important that surveillance is maximized in these areas. This article will review the embryology and gross anatomy of lymph nodes. However, the primary focus will be on the histological composition of these structures as well as clinically relevant points regarding lymph node function. At approximately 0.1 by 2.5 cm, the lymph node is a relatively small glandular structure that resembles a kidney-bean. It has a convexed surface that is penetrated by afferent lymph vessels. On the opposing side, there is a concavity that is penetrated by the supplying artery, vein and nerve and also allows exit of efferent lymphatic vessels. This concavity is known as the hilum of the lymph node. They are suspended in loose connective tissue that follows the large vasculature. Active lymph node. Stain: hematoxylin and eosin. Medium magnification. Lymph nodes are encapsulated by dense connective tissue comprised of elastin and collagen fibres along with interspersed fibroblasts. The convexed surface of the lymph node is pierced by numerous afferent lymph vessels. They extend to the deeper areas of the lymph node by way of the trabecular extensions of the cortex. As the trabeculae penetrate the lymph node, they continue as reticulin fibrils (type III collagen) that offer additional structural support to the gland. Trabeculae (histological slide) Cross sectional analysis of a lymph node reveals that it is subdivided into three regions: The outermost layer is the cortex. It is made up of a subcapsular sinus, cortical sinus and lymphoid nodules. The subcapsular sinus is the first space that lymph fluid from the afferent channels enters within the node. The fluid then travels from here to the cortical sinuses; which are branches of the subcapsular sinus. The cortical sinuses are also known as trabecular sinuses because they travel along the trabecular network within the lymph node. Cortex (histological slide) The endothelium of the trabecular sinuses are perforated by dendritic processes as well as reticulin fibres. Antigen presenting cells (APCs), circulating antigen, and lymphocytes flowing within the lymph can access the lymphatic tissue within the nodes through the disrupted endothelium. The cortical layer also has relatively large aggregates of helper T – lymphocytes and rapidly dividing B – lymphocytes in the peripheral part of the lymph nodes. Although both T cells and B cells are present in the cortex, B cells are more abundant than T cells are in this region. These lymphoid nodules are situated around the branched, interlacing extensions of the follicular dendritic cells (FDCs). The nodules may or may not have a germinal centre depending on if it is a primary or secondary follicle. Primary follicle (histological slide) Histological staining of lymph node samples are strongly influenced by the amount of antigen the cells of the lymph node are exposed to. Additionally, the number of cells within the node as well as the distinct separation of the cords is also influenced by antigenic exposure. As a result, the primary follicle is comprised of small dormant lymphocytes throughout, while the secondary follicle has a heterogeneous collection of large B lymphocytes that have already been activated by inciting antigens. Primary follicles absorb less histological stains then secondary follicles. This is likely due to fewer cells in the primary follicle when compared to the secondary follicle. Secondary follicle (histological slide) The germinal centre can be further subdivided into a dark zone, light zone and a mantle zone. Each zone facilitates different aspects of B cell affinity maturation. In the peripherally aspect of the germinal centre, quiescent B cells are found in the mantle zone. These cells are characterized by intense basophilic staining, small cytoplasmic volume and a heterochromatic nucleus. Other cells in the mantle zone include follicular dendritic cells as well as the occasional helper T lymphocyte and macrophages. The fate of B cells in the mantle zone can go one of two ways. These cells either remain in the lymph node and mature into antibody secreting plasma cells and remain in the lymph node, or they transform into memory B cells that re-enter the systemic circulation. Germinal center (histological slide) The other two zones of the germinal centre are the light zone and dark zone. The light zone contains centrocytes that interact with follicular dendritic cells that express intact antigen on their surface. Centrocytes with high affinity binding to the follicular dendritic cell antigen will persist, while those with weak binding undergoes apoptosis. While resident macrophages help to clean up apoptotic B cells, helper T cells support the remaining B cells and foster the class switching phase of the cellular maturity. Centrocytes (histological slide) In the dark zone of the germinal centre, the centroblasts are highly mitotic and have a strong likelihood of producing mutated antibodies. These are the source cells for the light zone. Centroblasts (histological slide) Deep to the cortical layer is the paracortex. Its margins blend with the superficial cortex and deep medulla. The principal distinguishing features are the absence of lymphoid nodules and the large number of T lymphocytes (both cluster of differentiation 4 and 8 positive T cells [CD4+ and CD8+]) within the stroma of the paracortex. Paracortex (histological slide) The paracortex also has unique venules known as high endothelial venules (HEVs). Most of the lymphocytes that enter the lymph node do so via these channels. They are made up of cuboidal endothelium that is fitted apically with integrins and glycoproteins. Both these surface markers allow unimpeded passage of lymphocytes (i.e. diapedesis) from the percolating blood into the lymph node. These specialized vessels are also present in the mucosa associated lymphatic tissue distributed throughout the gastrointestinal tract. However, they are at their highest level of development within the lymph nodes. The deepest layer of the lymph node is the medulla. It is subdivided functionally and histologically into two other regions; which are the medullary cords and sinuses. The cords are populated by plasma cells, as well as B – cells and T – cells. The cells are arranged in cord-like projections extending centrally from the paracortex. Medulla (histological slide) Interlacing between the cords are distended areas lined by discontinuous endothelium. The luminal surface of the sinuses also contains a vast network of reticular cell processes. They act as the final point of filtration of circulating lymph. The medullary sinuses are the terminal continuations of the peripherally located cortical sinuses. They eventually culminate at the hilum of the lymph node to form efferent lymphatic vessels. Medullary sinuses (histological slide) Lymph vessels are lined by a single layer of squamous endothelium. They are fitted with valves that promotes unidirectional flow of lymph from the afferent lymph vessels to the lymph node and then to the efferent lymph vessels. The afferent lymph channels bring lymph with either free floating or complement bound antigen into the subcapsular space. Of note, the subcapsular space extends around the entire lymph node except at the hilum. The lymph then flows through the cortical sinuses. Efferent lymphatic vessels (histological slide) The arterial supply of the lymph nodes is derived from the vessels they encircle within their respective regions. For example, the axillary lymph nodes are perfused by arterial branches of the axillary artery. Each lymphatic artery gains access to the node by way of the hilum. As the vessel penetrates this area, it gives off a myriad of straight branches within the medulla that also arborize to supply areas that are distal to the vessels. Hilum of the lymph node (histological slide) In the cortex, the straight arteries branch off into arterioles that form tightly packed anastomosing networks. The capillaries are more commonly seen around the germinal centres, where there are usually less arterioles. Capillaries also become more numerous when there is antigenic stimulation of the lymph node. The arterioles and capillaries then return to similarly numerous anastomosing networks of venules and veins. They tend to follow the course of the arteries and arterioles in the opposite direction. Eventually the veins leave the node via the same point of entrance of the arteries (the hilum). A few weeks after the initiation of cardiovascular development (third week of gestation), the lymphatic system begins to take shape (sixth week of gestation). Numerous studies dating from 1995 to 2002 identified vascular endothelial growth factor receptor 3 (VEGFR 3), vascular endothelial growth factor C (VEGF C) and prospero homebox 1 (PROX1) as integral components in the development of lymphatic vessels. While VEGFR 3 and VEGF C are essential for vascular differentiation from mesenchyme, the presence of PROX1 is imperative for lymphatic endothelial cell differentiation. Definitive confirmation of the venous derivative theory postulated in 1902 was recently confirmed in 2007. One theory proposed that outpouchings of the venous endothelium (i.e. diverticulae) result in the formation of lymphatic sacs. Another school of thought proposes that the lymphatic system, like the rest of the vascular network, originates from cells arising from the mesenchymal layer and develops separately from the veins. The former theory, proposed in 1909, is the most widely accepted concept. The lymph sacs develop adjacent to the primitive venous network. The lymph sacs identified at the early stage of development are paired (jugular and posterior lymph sacs) and unpaired lymph sacs (retroperitoneal and cisterna chyli). Jugular lymphatic sacs form around the superior cardinal veins (precursors to the jugular veins). There are axillary lymphatic sacs that develop around the right subclavian vein. On the left side, the thoracic duct forms on either side of the left brachiocephalic vein. There are also lumbar and iliac sacs forming caudally. The lumbo-iliac, jugular and axillary sacs are bilateral structures. Cisterna chyli The deep cervical nodes are the first to develop from its lymphatic sac. It is followed by the axillary, parasternal, and mediastinal lymph nodes; which also arise from their respective lymphatic sacs. The retroperitoneal, lumbo-iliac and inguinal nodes are derived from the lumbo-iliac lymphatic sacs. The differentiation from sac to nodes occurs when the sacs are invaded and segmented by adjacent mesenchymal cells. Axillary lymph nodes The mesenchyme also forms the lymph node capsule

and connective tissue stroma. More specifically, it gives rise to the fibroblastic reticular cells that produce the connective tissue stroma as well as its extracellular matrix. Eventually lymphoid tissue inducer cells that promote the development of lymphoid tissue within the lymph node are formed. There is subsequent invasion of the lymph nodes by thymic lymphocytes either shortly before, or during the antenatal period,. These T lymphocytes tend to migrate into the deeper cortex while the B lymphocytes migrate to the outer cortex of the lymphatic nodules. Certain groups of lymph nodes are responsible for draining lymph from particular regions of the body. For example, lymphatic fluid from the lower limbs and perineal area will drain to the inguinal lymph nodes. Keep in mind that this lymph may contain pathogenic antigen from potential invaders. This antigenic stimulation can result in local enlargement of lymph nodes draining that particular region. The antigenic stimulation may also lead to changes not only in the size of the nodes, but also their consistency. This process is known as lymphadenopathy; it is also applicable to any disease process that affects the reticuloendothelial system. Lymph node enlargement is considered significant based on how large the nodes get. A general rule of thumb states that any enlargement greater than 1 cm of more than one lymph node is considered lymphadenopathy. However, some clinicians say that enlargement should be related to the region the nodes are located in. In other words: Cervical lymph nodes greater than 2 cm are considered significant Axillary lymph nodes greater than 1 cm are considered significant Inguinal lymph nodes greater than 1.5 cm are considered significant Lymphadenopathy can be further classified as local or generalized. If swollen nodes are only seen in one region (i.e. cervical or axillary) then it is local. But if swollen nodes are found in more than one areas (i.e. epitrochlear and cervical) then it is generalized. Typically, swollen lymph nodes are reflective of self-limiting viral or bacterial infections in children. However, there are other non-benign conditions that may also result in lymphadenopathy. Furthermore, in adults, it is unlikely for lymph nodes to be found in a quiescent state as past exposure may incite long standing morphological changes within the nodes. Therefore it is important to differentiate between an ongoing disease process and a past exposure. Nevertheless, the swollen glands should not be ignored. Lymphadenopathy should not be confused with lymphadenitis. The latter refers to an acute inflammatory process within the lymph node. While this condition also presents with lymph node enlargement, it is characterized by painful, indurated lymph nodes. This is in contrast to lymphadenopathy where the lymph nodes are painless on palpation and are not erythematous. Clinicians can use clues from lymph node enlargement to determine where the underlying insult is occurring. Palpating lymph nodes is a clinical skill that is relatively easy to master and should not be neglected. Palpation of lymph nodes is particularly important during the general examination of a patient, as well as during examination of specific systems such as the respiratory system and abdominal exam. Common areas to palpate include the epitrochlear, axillary, cervical, pre and postauricular, occipital, submandibular and submental nodes. Inguinal nodes may also be palpated but the popliteal nodes are difficult to appreciate. In addition to the size and location of the nodes, the clinician should also note whether or not these nodes are tender, indurated and febrile as this points to a clinical diagnosis of lymphadenitis. Lymph nodes that are immobile, clumped together, and firm are more than likely (but not exclusively) a sign of malignancy than those that were described earlier. The list of aetiological factors leading to lymphadenopathy is quite eclectic. The can be generally classified based on whether or not the lymphadenopathy is generalized (systemic) or regional (localized). Below is a truncated list of the causes of lymphadenopathy: Generalized Infections Viral Bacterial Parasitic Autoimmune & Hypersensitivity disorders Systemic lupus erythematosus Drug reactions Storage disorders Neoplasms Regional (specifically those that are palpable) Cervical Upper respiratory tract infections Infectious mononucleosis Leukaemias Lymphomas Submaxillary and Submental Occipital Tinea capitis Rubella Roseola Pre-auricular Cutaneous infection Catscratch disease Supraclavicular Axillary Immunization reaction Non-neoplastic lesions of the breast Mastitis Brucellosis Lymphoma Inguinal Insect bites Sexually transmitted infections In cases where lymphadenopathy is believed to be a result of a non-benign process, then surgical excision of the node may be warranted. Lymphadenectomy is a diagnostic and therapeutic approach to managing patients with malignancies. It is often done in addition to removing a primary tumor. Recall that a hallmark feature of cancers is their ability to metastasize. If these malignant cells gain access to the circulating lymph then they can spread throughout the body and become lodged in the lymph nodes. This will result in the formation of a secondary site from which these nodes can spread. Therefore, clinicians use this information as a part of staging the malignancy. The stage of the malignancy also influences the mode of treatment that would be best for the patient. Lymphedema is a disorder of the lymphatic system characterized by swelling of the limbs as a result of disrupted lymphatic pathways. The etiology of lymphedema can be primary due to lymphatic hypoplasia which is inherited, or secondary due to obstruction or disruption of lymphatic vessels. Symptoms and signs of this condition are brawny, fibrous, nonpitting edema in one or more limbs diagnosed by physical examination. Primary lymphedema is uncommon and occurs as a result of hypoplastic lymphatic channels. The clinical presentation can vary in phenotype and patient age. There are also associations with lymphedema and other genetic disorders such as Turner syndrome. The causes of secondary lymphedema are more common and very often misdiagnosed. They are multicausal and can occur after surgery including lymph node dissection (typically breast cancer), radiation therapy, after trauma, lymphatic obstruction by a tumor, venous insufficiency, or infectious diseases such as lymphatic filariasis. Other than swelling, these patients may develop skin changes that include hyperkeratosis or hyperpigmentation. Further complications may lead to the development of elephantiasis (extreme hyperkeratosis that looks like elephant skin) or lymphangitis (acute bacterial infection of lymphatic vessels). Lymphedema is diagnosed by a good physical examination and additional tests with CT and MRI. The treatment of primary lymphedema is surgical including reconstruction while the treatment of secondary lymphedema means management of the cause. To mobilize fluid in lymphedema, physicians/patients may employ complex decongestive physiotherapy (CDPT). This involves a specific massage technique, skincare, and carefully fitted elastic compression garments. When applied correctly, CDPT dramatically increases lymphatic transport, delays the development of interstitial fibrosis, and improves symptoms. All content published on Kenhub is reviewed by medical and anatomy experts. The information we provide is grounded on academic literature and peer-reviewed research. Kenhub does not provide medical advice. You can learn more about our content creation and review standards by reading our content quality guidelines. References: Bailey, R. and Weiss, L. (1975). Light and electron microscopic studies of postcapillary venules in developing human fetal lymph nodes. American Journal of Anatomy, 143(1), pp.43-57. Butler, M., Isogai, S. and Weinstein, B. (2009). Lymphatic development. Birth Defects Research Part C: Embryo Today: Reviews, 87(3), pp.222-231. Dhar, A. (2017). Lymphadenitis - Dermatologic Disorders - MSD Manual Professional Edition. [online] MSD Manual Professional Edition. Available at: [Accessed 12 Sep. 2017]. Douketis, J. (2017). Lymphadenopathy - Cardiovascular Disorders - MSD Manual Professional Edition. [online] MSD Manual Professional Edition. Available at: [Accessed 12 Sep. 2017]. Gray, H. and Standring, S. (2009). Gray's anatomy. 40th ed. [Edinburgh u.a.]: Churchill Livingstone Elsevier. Kanwar, V. and Sills, R. (2017). 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Author, review and layout: Lorenzo Crumbie Uruj Zehra Adrian Rad Illustrators: Lymph node (histological slide) - Smart In Media Cisterna chyli - Begoña Rodriguez Axillary lymph nodes - Begoña Rodriguez Trabeculae (histological slide) - Smart In Media Cortex (histological slide) - Smart In Media Primary follicle (histological slide) - Smart In Media Secondary follicle (histological slide) - Smart In Media Germinal center (histological slide) - Smart In Media Centrocytes (histological slide) - Smart In Media Centroblasts (histological slide) - Smart In Media Paracortex (histological slide) - Smart In Media Medulla (histological slide) - Smart In Media Medullary sinuses (histological slide) - Smart In Media Efferent lymphatic vessels (histological slide) - Smart In Media Hilum of the lymph node (histological slide) - Smart In Media Histology of lymph nodes: want to learn more about it? 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